THE CHEMICAL BASIS OF ALLERGY

Working Hypothesis

Allergy or the hyperactivity of any cell function such as secretion, contraction, generation of nerve impulses, or cell division, not under physiological control, is the result of the photosensitizing action of a fluorescent substance adsorbed into the colloidal surfaces of the functional mechanism concerned, whereby the fluorescent substance directs the energy liberated by the usual exothermic reactions going on in the cell into the functional mechanism adsorbing it, and here this energy activates or accelerates the chemical reactions of this function. Thus the hypersecretion of hay fever, the spasms of asthma, or the increased cell divisions of neoplasia are instituted and maintained, and in the same manner other allergic activities are brought about (15). Most of the diverted energy is evolved in the hydrolysis of glucose to lactic acid, amounting to 270 calories per gram of lactic acid produced. Conversion of necrobiotic rays to mitogenetic rays through fluorescence is an important possibility in the gumma, leproma, and tubercle. Very little energy need be diverted to maintain certain forms of allergy, especially an allergy of the nervous system. Here one cell group continuously generating impulses, which traverse a train of neurones associated in one concept, establishes a fixed idea, a second personality, a neurosis, or a delusion of insanity. In the same way the activation of viruses is also made possible.

Fluorescent substances (1) are able to absorb energy either of radiation of certain wave length or the energy

liberated by exothermic chemical reactions in progress in the medium where they exist. In so doing they become a second system, which, owing to their limited storage capacity, reverts to their original state immediately giving up this energy, either as radiation of lower frequency or as chemical energy without degradation, if the fluorescent substance happens to be held intimately adsorbed into the surfaces of a reactive substance capable of serving as an acceptor of this energy. In this case, the energy received activates or accelerates the chemical processes of the acceptor. A fluorescent substance behaving in this way is called a photosensitizing catalyst. In order to serve in such capacity, both it and the acceptor must be able to absorb energy in the same spectrum ranges at a different range from that at which sensitization takes place, at which latter range only the sensitizer can absorb energy. The positions of atomic groups with unsaturated valences, with reference to the most reactive atoms of the molecule, determine the spectrum sensative regions characterizing the substance. In the case of the carcinogenic anthracene derivatives, (2) (3) the '9' and '10' 'meso-positioned' carbon atoms are most reactive and oxidize readily to keto groups. On this basis it happens that of the four different dibenzanthracenes only that with benzene rings occupying the 1, 2, and 5, 6, positions possesses correct spectrum absorption and emission qualities to be moderately carcinogenic. The 3: 4 benzopyrene molecule and cholanthrenes with appropriate meso-substitution are more and more energetically carcinogenic as their spectrum characteristics, or that of their quinones, coincide better with those of the mitogenetic mechanism of animal cells. One is tempted to predict that radiation of the living cell with their energy qual-

ities will result in hyperplasia. The crucial matter is that the fluorescent substance can take up energy evolved in exothermic reactions and be so changed that it emits this energy as its own characteristic emission spectrum. Thus the specificity of absorption of both sensitizer and acceptor exist at a different energy level from that at which the sensitizer absorbs the activating energy it passes on to the acceptor. To exemplify such photoactivity one might mention the "phosphorescence" of decaying vegetable matter, the light of the firefly, wereby excess energy of rapid muscle contraction is emitted as light which otherwise would accumulate as heat,* and the "cold light" displays where a fluorescent substance takes up energy from an oxidation reaction and emits it as light, also the sensitization of the gelatin-silver-salt complex of the photographic plate to red light by adsorbed fluorescent dyes, or the sensitization of Siloxen to the longer wave lengths by adsorbed rhodamine, and the acceleration of Siloxen reactions, because of its adsorbed oxy-compounds, to the chemical energy liberated as the reaction progresses. In this way in the allergic cell the fluorescent agent also is affected by this energy and as will be seen, the chemical resistance of the system it involves is removed so that it, itself, mediates a chain reaction using lactic acid and oxygen as reactants to evolve energy which also contributes to the allergy. So in two ways, energy for producing and activating the "hot" molecules of the carrier, which should continue the oxidation process beyond lactic acid, is removed. When the fluorescent substance behaves in this way, it serves as a negative catalyst to the oxidation mechanism. Since it re-

*This is my own explanation which I must admit has not been studied experimentally.

moves the energy ordinarily absorbable by, and necessary to, the production of the carrier of the normal oxidation chain, it must contain the same structural groups as the normal carrier. They are both subject to the same chemical reactions, therefore, and absorb energy in the same ranges competitively. This point is employed in building the chemical substances used to destroy the photochemic fluorescent sensitizer of allergy, the negative catalyst, that prevents the production of the carrier of the normal oxidation chain in the pathogenesis of cancer, and destroys resistance to infection.

Since fluorescence depends upon the unsaturated valences between carbon atoms, between carbon and nitrogen, and between carbon and oxygen atoms, and since, according to our views, all forms of allergy depend upon this fluorescence, and the "carrier" powers of the carbonyl group, only one substance, so constructed that it can saturate these valences, is required to overcome all forms of allergy, even including the allergy of the plastic system, neoplasia, and also to destroy virus activity.

Inasmuch as catalysts are active in low concentration, it is important that the reaction by which the fluorescence is removed should bring about a complete removal of every trace of the fluorescent structure. This cannot be accomplished by a reversible reaction such as enzymes mediate. Only a reaction chain in which the fluorescent substance serves as one of the reactants and the treatment measure is the carrier of the reaction could be expected to succeed completely. A brief statement on the nature of chain reactions (1) will explain this point. Their characteristic features are that the reaction is mediated by a "carrier", an activated mole-

cule or atom. This carrier renders the fresh reactant molecules active. The carrier once formed establishes a cycle of consecutive reactions which reproduce the carrier and produce the resultants as by-products of the cycle. High quantum yield results. The chain can only be ended by complete conversion of the reactants or by a destruction of the carrier, and the latter can only be done by processes other than those that maintain the chain. Hence, the last survivor of the chain is normally the carrier which, in the case at hand, may remain adsorbed in the colloids any length of time to reestablish the chain if fresh reactants ever enter the field again. The process then ends in immunity which, I have clinical observations that indicate, may be passed on to the next generation.

Since the anærobic oxidation chain is interrupted at lactic acid production (4) in the pathogenesis of malignancy and must be restored, the corrective measure to be preferred is that structure which reestablishes the ærobic oxidation of sugar and lactic acid and also engages the fluorescent groups in a vigorous oxidation chain reaction. Hence the unsaturated groups exhibiting the fluorescence and activated oxygen liberated in the body are employed as the reactants. The carriers chosen are the structure O=C=C=O, ("Glyoxylide") or less advantageously O=C=C=C=O ("Malonide"), the internal anhydrides of glyoxylic and malonic acids, ketenes and the peroxides of formaldehyde which functions in the same way. They may be grouped because of similar structure under the name "Ketenones." They appear to serve as the carriers of the normal oxidation chain and are employed therapeutically in loose combination with protective molecules or as precursors (peroxides of form-

aldehyde) from which they are liberated in active form in the body. To mediate the oxidation of the fluorescent pathogenic units they behave as photosensitizing catalysts and activate the union of peroxide oxygen and the unsaturated valences of the fluorescent atoms. The determination of their "carrier" structure depends upon an original interpretation of sugar oxidation.

It is based in part upon the influence of the carbonyl group in determining hydrogen shift between three carbon atoms where an ethylene linkage is envolved, and upon its tendancy to favor formation of an ethylene union between its adjacent alpha and beta carbon atoms. We may thus picture the production of a double bond able to add peroxide oxygen and subsequently split to two carbonyl groups. It appears to me that the degradation of fatty acids two carbons at a time is accomplished in this way and that the conversion of fats to sugars and sugars to fats is mediated by the same phenomenon. It is in these changes I believe, that the iodine of the thyroid and parathyroid secretions catalytically serve the oxidation mechanism, possibly through an oxide of iodine, and that the wasting of tissues by their excessive function is accomplished.

Our catalysts accomplish a cyclic inhibition and hastening of catalase activity either through a polymerization of, or a union with catalase, from which catalase is again regenerated. Various peroxides exhibit this activity when studied spectrographically.

The action of our catalysts may be interpreted as the activation of oxygen through their free valences preliminary to behaving as peroxides in one stage of their activity.